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Possibility of Polymicrobial Synergy and Dysbiosis of Periodonto Pathogens in the Oral Squamous Cell Carcinoma Tumour Micro Environment

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Editorial

The role of infection associated inflammation in oral cancer gained much attention and at least 15 % of oral cancer is suspected to be associated with chronic inflammation [1-3]. Poor oral hygiene has emerged as an independent risk factor for Oral Squamous Cell Carcinoma (OSCC) substantiated by evidence of recent past research [4-6]. Poor oral hygiene may cause periodontitis [7]. In several studies, periodontitis reported to be associated with increased risk of OSCC [8-12]. Periodontitis is an inflammatory disease of the periodontium caused by combined action of polymicrobial communities [13]. Carcinogenic attributes of two periodontopathogenic bacteria *Fusobacterium nucleatum* [3] and *Porphyromonas gingivalis* [3] have been revealed by in vivo and in vitro studies, especially these two major periodonto pathogens reported to be promoted tumour progression [3]. Furthermore, these two well established periodonto pathogens have been coupled with pancreatic and colorectal (CRC) cancers respectively [3]. These two and several other periodontopathogens were detected in saliva, surface swabs and tumour tissues of oral cancer patients [3].

Contemporary advancements in the next generation sequencing technologies and 'omics methods'; metagenomics, metatranscriptomics, meta proteomics and metabolomics which overcame the inherent limitations in conventional culture techniques and molecular techniques such as PCR and DNA – DNA hybridization have provided an extra ordinary opportunity to understand microbial ecosystems [3]. Furthermore, to appreciate the versatility of microbes to adopt to harsh environments based on the stability, dynamic equilibrium and self-organizing ability of micro-ecosystems. Thus, 'Hypothetical Models' have been proposed to explain the progression of inflammatory poly microbial diseases in the oral cavity. Especially, contribution of low abundance microbial species in 'poly microbial infections' which can act synergistically with over presented marker genera which is termed as 'polymicrobial synergy' [13,14,15] to maintain a persistent inflammation in the oral cavity [13]. The term "keystone" has been introduced in the ecological literature to characterize species whose effects on their communities are disproportionately large, relative to their abundance and which are thought to form the "keystone" of the community's structure [13,14,15]. The 'Keystone Pathogen Hypothesis' holds that certain low abundance microbial pathogens could orchestrate inflammatory diseases by remodeling a normally benign microbiota into a 'dysbiotic' one. Dysbiosis is defined as a state of imbalance that is characterized by compositional and functional changes of bacteria compared with the proportion of healthy state [3,14,15], by dominance of 'key stone' pathogens in the disease status [3,13]. **Key-Stone Pathogen** [14] mediated **Polymicrobial Synergy and Dysbiosis** (PSD) model [13] have been developed and validated to explain the progression of the disease in periodontitis based on alpha-diversity and species composition. Epidemiological evidence confirms the fact that, oral microbiome appears as a cofactor in the initiation and progression of oral cancer [3]. Against this back drop, there is a possibility of polymicrobial synergy and dysbiosis mediated by periodonto pathogens in the OSCC tumour microenvironment. Metagenomic and metatranscriptomic studies based on the large cohort study design is much warranted to explore the possible association of polymicrobial synergy and dysbiosis mediated by periodontopathogens in the OSCC tumour micro environment.

This invited editorial highlights the importance of investigating the possibility of polymicrobial synergy and dysbiosis of periodonto pathogens in the Oral Squamous Cell Carcinoma tumour micro environment. This will provide the rationale of improvement of oral hygiene status especially treatment for chronic periodontitis of OSCC patients after main treatment modalities; surgery, chemotherapy and radiotherapy.

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